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Figures 2A-<sup>2</sup>D are sections depicting staining for the presence of integrin linked kinase, showing a correlation with psoriatic disease progression in epidermal keratinocytes and dermal vascular endothelium.

Figure 3 depicts the anti-inflammatory activity of the anti-ILK compound MC-5 on edema.

#### DETAILED DESCRIPTION OF THE EMBODIMENT

- [13] Inflammatory disorders, including autoimmune diseases, are treated by administration of inhibitors of integrin linked kinase (ILK). Such disorders and diseases include, but are not limited to, psoriasis, rheumatoid arthritis, multiple sclerosis, scleroderma, systemic lupus erythematosus, Sjögren's syndrome, atopic dermatitis, asthma, and allergy. Cutaneous disorders are of particular interest. Target cells susceptible to the treatment include cells involved in instigating autoimmune reactions as well as those suffering or responding from the effects of autoimmune attack or inflammatory events.

#### ILK MODULATING AGENTS

- [14] ILK is a 59 kDa serine/threonine kinase that associates with the cytoplasmic tails of  $\beta 1$  and  $\beta 3$  integrins. The enzymatic activity for ILK is modulated by the interaction of cells with the extracellular matrix component fibronectin, integrin clustering and a number of growth factors. Because of its intimate association with a wide variety of signaling pathways that have been directly or indirectly implicated in various pathological processes, ILK represents a therapeutic target for a variety clinical conditions including angiogenesis, cancer, inflammation and autoimmunity. The genetic sequence of human ILK is disclosed in U.S. Patent nos. 6,013,782; and 6,001,622, herein incorporated by reference.

- [15] Overexpression of ILK results in a downregulation of E-cadherin expression, formation of a complex between  $\beta$ -catenin and the HMG transcription factor, LEF-1, translocation of  $\beta$ -catenin to the nucleus, and transcriptional activation by this LEF-1/ $\beta$ -catenin complex. LEF-1 protein expression is rapidly modulated by cell detachment from the extracellular matrix, and LEF-1 protein levels are constitutively upregulated upon ILK overexpression. These effects are specific for ILK.

- [16] Agents that block ILK activity are used in the treatment of inflammatory disease, including psoriasis. Numerous agents are useful in reducing ILK activity, including agents that directly modulate ILK expression, e.g. anti-sense specific for ILK, ILK specific antibodies and analogs thereof, small organic molecules that block ILK catalytic or binding activity, etc.; and agents that affect ILK activity through direct or indirect modulation of [PtdIns(3,4,5) $P_3$ ] levels in